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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Neurology and the Nervous System of Man

ALEX J. ARIEFF, B.S., M.D.
Chicago, Illinois

The knowledge of the structure and function of the nervous system of man has been derived chiefly from the study of man.

Since it is impossible to perform ablations of parts of the nervous system in man, many discoveries related to the function of the nervous system have been derived from such experiments as nature provides in disease and injury. Otherwise approximations and analogies have been made from studies of animals, particularly primates. Nevertheless, much has been learned from clinical observation coupled with pathology.

The neurologist singularly has been prominent in such studies and we who bear the mantle of distinguished forebears can look with pride at their accomplishment and be spurred to zealous effort for future endeavor.

I should like to refer to a quotation from Cushing at the opening of the Montreal Neurological Institute[1] in 1934:

"Psychiatry, to be sure, had been a recognized specialty longer than neurology . . . yet it had seemed clear to us that organic neurology supplied the only possible link to hold all interested parties together for their mutual advantage. Unquestionably, a proper foundation in organic neurology, in neuropathology, and physiology should be the starting point for psychiatrists as well as neurosurgeons, and while they once seemed far apart, a year's close association with Adolph Meyer in Baltimore had given one ample reason to know how profitable to both parties such a contact might be." And in the same address he quotes Charles Dana, who in 1913 in speaking of the "Future of Neurology" said, "It seems to me that neurology will, to a degree for a time, divide up into specialties of its own. There will be neurologists who are especially students of organic neurology; there will be functional neurologists; psychotherapeutic and psychoanalytic; electro-therapeutic; epileptic, glandular, neuroserological, and laboratory neurologists, but the definite fruit of their special work will become systematized and absorbed by the general neurologist, who will come into his own as a master of that highest branch of medical art which deals with the master tissue of the human body." . . .

"This prophecy, I believe, will prove true, but that neurosurgery was soon to be added to the subdivisions of neurology was unforeseen even by so clearsighted a man as Dr. Dana."

Although one uses and takes for granted the objective tests in doing a neurological examination, it must be recalled that most of the observations are not too old. Furthermore, the contributions in neuropathology and neurophysiology have been made for the most part by neurologists or practitioners with neurological interests who were strongly motivated by curiosity and zeal for research.

In examining the peripheral nervous system, a few names should be highlighted to refresh our memories. Sir Charles Bell of Edinburgh, an eminent neurological clinician, in 1811 privately printed a dissertation on the function of the anterior root.[2, 24]

"On laying bare the roots of the spinal nerves, I found that I could cut across the posterior fasciculus of nerves which took its origin from the posterior portion of the spinal marrow without convulsing the muscles of the back; but that on touching the anterior fasciculus with the point of a knife, the muscles of the back were immediately convulsed. . . ."

Francois Magendie[2] was a physician to La Salpetriere in 1826, but directed his atten-

From the Department of Nervous and Mental Diseases, Northwestern University Medical School, Chicago, Illinois.

Presidental address, read at the Chicago Neurological Society, April 11, 1950.

tion to experimental physiology. He published the first physiology journal in French and in the second volume in 1882 describes cutting the posterior roots in six-week-old puppies noting that the limb did not move to pin prick but was able to move voluntarily.

"I began to think it probable that posterior roots might have different functions from anterior roots and that they were more particularly destined for sensation."

In another experiment, Magendie finally cut anterior roots where they pierced the dura, "the member was completely immovable and flaccid, at the same time preserving an unequivocal sensibility." When both roots were cut at one time, "there ensued absolute loss both of sensibility and motion."

Herbert Mayo[2] took the next step in 1822 by describing the function of motor power of the VIIth cranial nerve and facial sensation by way of the Vth cranial nerve.

August Waller of Faversham,[3,4] Kent, England, a physiologist, a poor teacher, but a great researcher, in 1850 laid down the principles of degeneration of neurones.

"If glossopharyngeal and hypoglossal nerves be severed, the outer segment containing the axis cylinder cut off from the cells, will undergo degeneration while the central stump will remain relatively intact for a long period of time."

He inferred that nerve cells nourish the nerve fibers. By the same method, if anterior root is severed, the degenerative changes indicate nutritive centers of motor fibers in the spinal cord, while in section of the posterior sensory roots they are seen to lie in the posterior root ganglia. For this he won the Montyon Prize of the French Academy of Science, 2,000 francs, in 1856.

Thus, the pathways to and from the spinal cord and laws of degeneration were described.

After Faraday[5] in 1832 described galvanic and magnetic induction, electricity expanded the science of neurology. In 1848, DuBois Reymond[6] published his treatise on animal electricity. This laid the foundation for further clinical contributions.

Guillamme Benjamin Armand Duchenne,[7,31] a neurologist (1806-1875), tried to establish himself in Paris and offered his services to the various hospitals only to have doors slam in his face. He kept on trying and finally was accepted and utilized for his famous studies. He found that he could isolate and stimulate various nerves and muscles by electrodes placed on the skin. He studied the muscular system intensively, founding and popularizing electrophysiology and diagnosis.

Although Jacob Heine,[31,8] a neurologist, described infantile poliomyelitis in 1840, it was Duchenne[8] in 1855 who placed the lesion in the spinal cord. And in 1864 Charcot[8] showed the relationship of the muscular atrophy to the spinal cord lesion. Duchenne[8] then with Aran found the spinal cord lesions causing progressive muscular atrophy and separated the spinal atrophies from the dystrophies. In 1868 he described pseudohypertrophic muscular dystrophy. He also described glossolabial bulbar palsy.

Further contributions were made by the neurologist Wilhelm Erb[3,7] (1840-1921). His early studies were in pathology and internal medicine and his early association with Friedreich in 1867 influenced his life. Of his 237 publications, three-fourths were neurological. He introduced his methods of electrodiagnosis in 1868 and his book on electrotherapy was published in 1882. His description of "Reaction of Degeneration" still stands today with little improvement in spite of recent electronic advances.

Henry Head,[9] the famous neurologist, in the famous experiment on himself, in which he cut his own external radial cutaneous nerve and studied loss and then restoration of sensation is a classic. His study of the distribution of pain and tenderness in visceral disease in 1893 which corresponded to root ganglia of the spinal nerves gave us the "Head Zones." His other studies including sensation and its representation up into the brain are still a classic.

James Sherren[10] (1872-1945) started out before the mast, then entered medical school and became FRCS in 1900. As a busy surgeon he found time to do pioneer research work with Head on the peripheral nervous system, and in 1908 published a monograph "Injuries of Nerves and Their Treatment."

Lewis J. Pollock's[11] elaboration on sensory overlap cleared up many erroneous ideas in evaluation of recovery of peripheral injuries.

The Spinal Cord

Robert Whytt[2,3,11] (1715-1766), a neurologist of Edinburgh, approached the physiology of the nervous system in the modern spirit, extirpating known regions of the central nervous system and studying how animals reacted thereafter to various stimuli. In his book (1751) he established that the spinal cord is essential for reflex action and described the pupillary response to light, observing that destruction of the anterior corpora quadrigemina abolished the reaction. He showed that reflex action does not depend upon the integrity of the cord as a whole, but that an isolated segment suffices for its reaction.

Marshall Hall[2,3] (1790-1857), a neurologist, was also an indefatigable experimenter who introduced the concept that the spinal cord is a chain of segments whose functional units are separate reflex arcs. In 1833 he demonstrated cessation of strychnine convulsions after destruction of the spinal cord and that most reflexes were more readily elicited by stimulating appropriate end organs than through their bared nerve trunks. He also demonstrated tonic closure of the sphincters by reflex action. Of these reflexes he writes:

"Three things are plain from these observations: (1) that the nerves of sensibility are impressible in portions of an animal separated from the rest, in the head, in the upper part of the trunk, in the lower part of the trunk, (2) that motions similar to voluntary motions follow these impressions made upon the sentient nerves and (3) that the presence of spinal marrow is essential as the central and cementing link between the sentient and motor nerves. . . ."

Charles Brown-Séquard,[2,8,30] a neurologist with strong physiological interests (1817-1894), the son of an American sea captain and French mother, was born in Maritius. Because he was discouraged, like Claude Bernard, from becoming a poet, he took to medicine and was a roving pioneer with activities which rose and fell. He worked 18 hours a day, sleeping from 8 P.M. to 2 A.M.

In his experiments on transection of the spinal cord he found that transverse division of the lateral half of the cord produced loss of voluntary movement with "Hyperesthesia" (really meaning hyperreflexia) below the lesion, on the same side, and loss of sensation on the opposite side. On the other hand, a longitudinal section in the midline produced a loss of sensation corresponding to the length of the lesion without loss of voluntary movement. He wrote:

"It seems absolutely certain, from the above facts and reasoning, that there is no decussation of the voluntary motor fibers of the trunk and limbs above the crossing of the pyramids . . . so that wherever the lesion in a lateral half of the cerebrospinal axis may be—below, above, or at the level of the crossing of the pyramids—if it produces anesthesia, it is in the opposite side; while paralysis, in these three cases is either in the same or the opposite side, or in both sides."

Moritz Heinrich Romberg[3,8,31] (1795-1873) was the first systematic writer to present the viewpoint of the German physiologic school. He described such reflexes as the cremasteric in clinical diagnosis. Although at an earlier date Alfred Volkman[3] (1800-1877) had pointed out that after spinal cord injury, previously subliminal stimuli became adequate for the production of a response and consequently that higher neural levels must exert an inhibitory influence upon segmental reflexes. It was Romberg who described the reflex pattern after cord section and cerebral lesions:

"In cerebral paralysis the power of inducing movement by stimulus of mental impressions is entirely extinct or imperfect. . . . On the other hand, the capability of being excited by other stimuli continues and is even exalted. . . . If the volar surface is touched, especially with a hot or cold body, the hand and entire arm executes a rapid movement. . . . It will sometimes happen that in other reflex movements, e.g., respiration, yawning or coughing, the affected limb makes sympathetic movements, while the healthy limb remains at rest. . . ."

John Hughlings Jackson (1834-1911)[1] in his concepts of "evolution and dissolutions" agreed that some of the manifestations such as decerebrate rigidity were to be interpreted as due to abnormal degrees of stimulation or to enhancement of the reflex arc by the removal of inhibition.

He was greatly influenced by Jonathon Hutchinson and Brown-Séquard. His contributions in the cerebral area include focal epilepsy, called Jacksonian by Charcot, the levels of the nervous system and the concept of evolution and dissolution, e.g., the higher centers being refined and complex are inhibitory over the lower centers. Destructive lesions in the nervous system do not produce positive symptoms which are

really due to normal structures' acting released from influences of higher diseased centers. This he illustrated in aphasia, hemiplegia, post epileptic states and disorders of mentation. He published 300 papers including even a paper on the "Psychology of Joking." After Helmholtz introduced the ophthalmoscope, Jackson made ophthalmoscopy a science.

In the World War I description of mass reflexes after spinal injury were confirmed by the famous neurologists, Henry Head[9] and Gordon Holmes.

Jean Martin Charcot[3,7] who made his famous observations while at Salpetriere, attempted to correlate the purely physiological organization of the Romberg symptom complexes with pathology. Even at this age diagnosis consisted mainly of observation and palpation and the electrical stimulator. Later the ophthalmoscope and reflexes were of greater aid.

In speaking of bilateral paralysis of the upper extremities, Charcot differentiated clinically anterior root lesions from upper motor neurone disease. He also contrasted anterior horn disease (ventral gray column damage) from conduction system disease which he called myelitis. It was only later that the pyramidal tract was identified as responsible for hyperreflexia and the tract of inhibition.

"The controlling influence of the cerebrum over reflex acts is, moreover, placed in its true light when we consider what takes place after section of the cord." "But it is early shown that this influence is especially extoled through the antero-lateral fasciculi. Indeed the section may be such that sensibility is preserved and furthermore, one side of the cord only may be affected." . . . "The antero-lateral columns are, therefore, the conductors of voluntary stimuli . . . yet we shall see that pathological facts settle the question which physiological experiment leaves undecided."

In Charcot's 11th and 12th lectures he pointed out since the pyramidal tract undergoes degeneration in apoplexy and the patients lose voluntary control, this tract is responsible for volition. Among signs of degeneration he describes as ankle clonus and exaggerated tendon reflexes. The patellar reflex had been previously studied by Erb and Westphal.

Henry Bastian's[12] description of loss of reflexes and flaccidity in acute traumatic myelitis, although in part untrue and only temporary, is a "classic." He also described sensory centers to voluntary movements and muscular sense.

Babinski[3] reported his great discovery, a pathologic reflex, in twenty-eight short lines in 1896, and very little has been added to this since most of the other pathological reflexes are modifications.

Sherrington's[2] studies on the integration of the nervous system opened up a new era in neurology. Just a few contributions include localization of the knee jerk, somatic sensorimotor segmental innervation of the monkey, preliminary to study of the reflex functions of the spinal cord, reciprocal innervation and integration of reflexes. After the spinal cord he studied the higher centers.

In connection with Sherrington should be mentioned Rudolph Magnus'[2,13] (1875-1927) studies on posture which started in Sherrington's laboratory in 1908. These studies were aided by DeKleyn. Magnus was a physician who early became interested in pharmacology. He published over 300 papers including about 150 of his studies on posture. Among them were many papers on the sympathetic nervous system. G. G. J. Rademaker,[32] Dutch professor of neurology, one of the many who worked with Magnus, also contributed to studies of posture, labyrinthine reflexes and equilibrium. Lewis J. Pollock[33] and Loyal Davis furthered studies on posture when they described their anemic method of decerebration.

William Gowers,[12,14] who did much to further the clinical knowledge of the brain and spinal cord may be remembered for his descriptions of the ascending ventral spinocerebellar tract. With Horsley[20] he was the first to localize and have removed a cord tumor successfully. Another of Gower's contributions to hyperkinetic movements was when he called attention to a large group of cases which the American neurologist Hammond[31] called athetosis, and placed them in post hemiplegic disorders of movement (athetosis, choreic movements and tremors).

Cerebral Function

Gall and Spurzheim[3] were the first who brought out the importance of the gray matter of the cerebral cortex in general, but

it was not until 1870, when Gustav Fritsch[15] and Eduard Hitzig published their first memoir "On Electrical Excitability of the Brain," that a brilliant opening was made in furthering our knowledge of the brain. This made even more significant Broca's[16] famous paper of 1861 in which he ascribed the essential role of speech production to the left third frontal convolution.

Eduard Hitzig,[17] a neuropsychiatrist (February 1838-August 1907) born in Berlin, early showed his interest in neurophysiology. When he began his work there were no laboratories for warm blooded animals so his work was done in his home in a bedroom using a dressing table for operations. He was assisted by his wife and Fritsch. His first work was published in 1870 on "Electrical Excitability of the Brain." Later works included the subcortical structures. In 1874 Fritsch and Hitzig proved that ablation of the occipital cortex caused blindness. In 1875 he became Professor of Psychiatry and in that same year the Director of the Neuropsychiatric Clinic at the University of Halle. (He had to give up work in October, 1903, because of a severe eye infection.) He stated that psychology that did not have its roots in the natural sciences did not interest him.

Sir David Ferrier,[2] an English neurologist (1843-1928), verified the observations of Fritsch and Hitzig clinically, extended the knowledge of cortical and subcortical localization. "The experimenter can predict with certainty the result of stimulation of a given region." He determined effects of destruction of limited portions of the cerebral cortex. He allocated regions specially concerned with vision and hearing. He showed that hemiplegias and monoplegias resulting from the injured motor region of the ape were greater than in the dog. In 1881 at the International Congress of Medicine in London, Sir Charles Ballance relates that Charcot, upon seeing one of the hemiplegic monkeys exclaimed, "It is a patient!" In 1876 "The Functions of the Brain" was published.

Another distinguished English neurologist, mentioned before, was Henry C. Bastian.[12] After receiving his M. A. degree in 1866, he became a lecturer in anatomy, 1867, and Professor of Pathology in 1867. He was a great philosopher and honest analyst. His laboratory and consulting room were separated only by a curtain. His views on aphasia were classic. He described word deafness in 1869—five years before Wernicke. In 1888 his work on "Brain as an Organ of the Mind" describes the nervous system throughout the animal kingdom up to man including consciousness, instinct, and volition. As a biologist, a good portion of his later years were spent futilely proving abiogenesis. In mentioning aphasia, the neurologist Pierre Marie must not be forgotten. He was also responsible for work on cerebellar disease and arthropathies.

S. A. K. Wilson[19,31] (1878-1937) studied with Pierre Marie at Bicetre and attended the Babinski clinic at the old Pitié. He had early contacts with Gowers, Bastian, Horsley and Jackson. His studies on aphasia are classical. As a research fellow he worked on "Lenticular Degeneration" published in 1912 and now called Wilson's Disease. Among his modern views on neurology are included chapters on epilepsy, narcolepsy, striatospinal and corticospinal motor system, disorders of motility and muscle tone, pathological laughing and crying, dysaesthesias and Argyll Robertson's pupil.

When mentioning hyperkinaesias, it should be recalled that as early as 1817 James Parkinson[2,8] of Horton, Middlesex, a neurologist, recognized the pathology and signs of paralysis agitans. "Involuntary tremulous motion with lessened muscular power, with propensity to bend trunk forward and to pass from a walking to a running pace, the sense and intellect being unimpaired."

Herman Nothnagel,[8,20] (1841-1905) an internist in Vienna where he was Professor of Special Pathology and director of the clinic besides having a large private practice, contributed to localization in the brain of epilepsy and diseases of the nervous system after typhoid fever.

Foerster,[21,22] a neurologist who did his own surgery, opened up research on the projection fibers system. A landmark was established in neurophysiology when Foerster obtained repetitive effects such as a continual stream of enunciated words from stimulation of Broca's convolution and auditory hal-

lucinations of spoken words from stimulation of Wernicke's area in the temporal lobe. It has been remarked, "The study of the function of a cerebral lobe by the study of the remainder of the brain after the operation is like studying the functions of a violin by studying an orchestra after the removal of the violin." Since Foerster's work the functions of lobectomy are those postulated previously by the neurologists. Foerster also made profound investigations on the spinal cord, cause of pain, spastic and flaccid paresis, cutting posterior roots to eliminate pain and decrease spasticity.

A word should be said of electrophysiology with respect to electroencephalography. Hans Berger,[23] a neuropsychiatrist (1873-1941), is considered the father of clinical electroencephalography. He was Professor of Diseases of Neuropsychiatry in Jena from 1905 and published numerous papers on the brain, circulation, traumatic psychosis and brain localization.

Neurosurgery

Sir Victor Horsley is considered the father of neurosurgery.[1] In 1880 he was closely associated with Bastian. In 1882 as assistant professor of pathology at University College for six years he did physiological research with Schaefer and Beevor and made studies on localization in brain and pathology of epilepsy. He was early associated with neurologists in research in psychology and surgery of the nervous system. At thirty in 1886 as Surgeon to National Hospital, Queen's Square, he did eighteen operations on ten patients with one death. Aided by Gowers in 1887 he successfully removed a cord tumor.

Harvey Cushing[1] although not trained by Horsley really succeeded him. After graduation from Harvard in 1895, he assisted Halsted at Johns Hopkins and came under the spell of Osler. Because of his interest in Roentgen rays he studied the neurologic course of a patient with a gunshot wound of the spine. In 1900 he turned to the nervous system. In Kroenecker's laboratory in Berne he showed that blood pressure increased with increased intracranial tension. In Sherrington's laboratory with Grunbaum, he assisted in delimitation of motor cortex of anthropoids. Here he showed his artistic talent by drawings. In 1901 as orthopedic surgeon he operated on neurologic cases and his neurosurgical fame spread. He pioneered work on blood pressure and developed subtemporal decompression. He investigated the physiology of cerebrospinal fluid and the pituitary body. As Professor of Surgery at Harvard, he began his great school of neurosurgery, so that now there is scarcely any place in the world without one of his students.

Another American landmark in neurosurgery was begun when the neurologist, William Spiller, with W. Keen reported on resection of the Gasserian ganglion in 1898 and then Charles Frazier operated on Spiller's case of "Tic Douloreux" and successfully cut the sensory root in 1901.[24]

From that time knowledge has grown by leaps and bounds and the neurosurgeon has been of greatest importance. A recent example is Brickner's[34] intensive study of Dandy's case of bilateral frontal lobectomy.

Cyto Architecture

This realm is replete with basic neuropsychiatric contributions.

Alois Alzheimer (1864-1915)[1] and Franz Nissl are usually associated. Nissl actually opened the era of neurocytology and pathology by using alcohol as a fixative and staining with magenta red which was soon replaced by methylene blue. He stained the Nissl substances and studied the axone reaction after interruption. He initiated cyto architectures by demonstrating thalamic projection of cortical areas. With Weigert in 1885 he contributed to the staining of supporting tissues, blood elements, and blood vessels in brain pathology. He did epic work on cortical pathology in general paresis. He was a thorough clinician at Heidelberg, later Professor and Director of the Psychiatry Clinic. The daytime belonged to his patients and the nighttime to his research. In 1904 he became Professor of Psychiatry and Director of the Klinick in Munich. In the state asylum of Frankfort he met Alzheimer in 1839. They worked more or less together for 25 years. Their combined classic work was published from 1904 to 1918. Alzheimer's work in pathol-

ogy of the cerebral cortex culminated in the disease bearing his name. He recognized ganglion cell changes in various psychotic states.

Theodore Meynert,[25,26] a neuropsychiatrist (1833-92), who was active in Vienna is also considered the father of architectonics. He discovered the projection fibers, the commisural and association systems. Although Meynert was born in Dresden he was early established in Vienna. At thirty-two he started the study of the brain and outlined a project on the anatomy of the cerebral cortex as a carrier of conceptual functions and its relationship to sensory and motor functions. He actually laid the groundwork for Fritsch and Hitzig and Munk and even for the later works of Brodmann, Vogt and Economo. In 1870 he was made director of the first psychiatric clinic in Vienna, where, after having studied normal brain function, he went on to clinical material and contributed pathological psychiatric observations.

Constantin Von Monakow[1] (1853-1930) was never a good student except when he taught himself. After many attempts in various fields, he finally in 1872 entered the medical faculty in Zurich in a field in which he was not too interested. He became interested in pathologic anatomy under Ebert and became a psychiatric assistant to Hitzig in 1876. He showed great promise and was stimulated by Von Gudden in learning how to use the coarse microtome. He graduated and practiced only a short time. He became an assistant physician in the insane asylum of St. Parminsbergen, Switzerland. He spent seven years sectioning formalin fixed brains with a Gudden microtome. The brain was stained with chromic acid and held by a mixture of stearin, pigsfat and suet. He made various lesions in the brain and studied the anatomical changes. He demonstrated atrophy of external geniculate body in destruction of the occipital lobes. He demonstrated various anatomical projections. He published studies on cyclops, reduplication of spinal cord, microcephaly, aproxia, post hemiplegic disturbance of motility. He founded the German association of neurologists in 1909. Later his interest changed from aphasia, apraxia and agnosia to psychiatry and philosophy.

Neurone Theory

J. E. Purkinje,[20,31] a physician who held the Chair of Physiology and Pathology in Breslau described cerebellar cells in 1837 but it was Remak who discovered the axis cylinders and noted that they were made up of fine fibers. Rosenthal,[31] a pupil of Purkinje, introduced the medullary sheath in 1832. Schwann[31] described them in 1838. Hannover,[31] in 1840, stated that nerve fibers came from nerve cells. Remak[20] in 1855 discovered axis cylinders and pointed out that anterior born cells have but one axis cylinder. Waller in his studies on neurone degeneration strengthened the neurone theory in 1850. Gratiolet[31] showed that fibers come from the anterior horns. Golgi, Ranvier, Berlach, Weigert, and Marchi and Cajal by their histological studies and stains made important contributions to the neurone theory.[31]

Finally Wilhelm Waldeyer[27] (1836-1921) was awakened by Henle to a permanent interest in anatomy. In 1867 he held the Chair of Pathologic Anatomy in the University of Breslau. In his "Investigations in the Realm of Anatomy of the Central Nervous System" he disclosed and established that the so-called neurone possessed anatomic, genetic, functional and trophic nerve unity.

The neurologist, Camillo Golgi (1843-1926), was born in Corteno, an Alpine village and studied medicine in Pavia. Influenced greatly by Lombroso and the pessimism of obscure functions of the nervous system he studied closely the microscopic anatomy of the nervous system. In 1870-71 he was able to describe neroglia of the cerebral gray and white matter. In 1872 his laboratory, because of economic conditions, was his kitchen. Working by candle light he discovered his epoch making chromate of silver method. His work from then on was monumental. Golgi also did brilliant research in malaria. He had three passions, his wife, the laboratory and the University of Pavia. Firmly convinced of the importance of histology he retained the honorary direction of the hospital department so that his pupils might maintain bonds uniting science with the practical side of medicine.

Cajal (Santiago, Ramon Y., 1852-1934),[1]

neuropsychiatrically trained, was born in Petilla, Spain, and had a life which reads like a fairy tale full of adventure. A poor student, he was a "problem child" and took up one occupation (barbering, shoemaking) after another. Twice he almost died from severe illness. The morphology of bones studied with his father set him off to study medicine. In 1884 he was called to the Chair of Anatomy at Valencia and in 1887 he became Professor of Histology at Barcelona. He experimented on his own after discarding the Golgi stain and skillfully stained and illustrated his own preparations showing nerve fibers and cells. He had to demonstrate his stains and results personally and finally sold first Germany and then the world. His most striking preparations were of nerve fibers of the molecular layer of the cerebellum, the ascending and descending branches of sensory nerves, and the termination of retinal fibers in the optic lobes of birds. He described for the first time the true relation of the nerve fiber to the nerve cell with a clear description of the basket structures about the Purkinje cells. He developed many new stains and demonstrated structures never before seen. His endeavors in other fields of writing and photography are noteworthy. In 1906 with Golgi he received the Nobel Prize.

Vegetative Nervous System

In 1851 Claude Bernard[3, 24, 4] (1813-1878) the great physiologist explained vasomotor function of sympathetic nerves. In 1853 Waller simultaneously with Claude Bernard and Brown-Séquard discovered vasoconstrictors in the sympathetics and in 1858 Claude Bernard discovered the dilator nerves. Waller also had investigated trophic nerve cells and with Budge in 1851 traced sympathetics to the spinal cord and by stimulation experiments showed its action on the pupil and defined the "ciliospinal" region.

Felix Alfred Vulpian,[28] a physician to La Salpetriere, in 1860 was a professor of pathological anatomy and held the Chair of Comparative and Experimental Pathology in 1872. In an appointment to the Museum of Natural History he experimented on the nervous system. He described the nature and functions of the vasomotor nerves. Charcot called him a man of duty and work his supreme refuge. When Pasteur was attacked Vulpian supported him.

J. S. Jewell,[24] first president of the American Neurological Association, in 1875 in the opening pages of the Journal of Nervous and Mental Diseases began with a series of lectures in pathology of vasomotor nerves. He described personal clinical experiences in the Civil War, certain cervical sympathetic syndromes involving blod vessels, and referred to S. Weir Mitchell's similar cases; he explained the reflex arc involving the cord, discussed vascular tonus and parasympathetic activities even though he did not have structural facts of Gaskell and Langley. Jewell also described trophic function as related to neurogenic activity in skin, arthritic and sympathetic syndrome.

T. E. Clark[24] in 1876 described injury to superior and inferior cervical ganglia. S. Weir Mitchell[24] in 1863 described a new sympathetic peripheral nerve syndrome of war wounds called "Causalgia," and in 1872 described erythromelalgia. Bannister,[24] an early American neurologist, described cervical sympathetic function in 1879. Mills[24] wrote a review of "Sympathetic Nervous Disease" in 1880. Clevenger[24] wrote on the "Plan of Cerebrospinal System" in 1880 Van Gieson,[24] supported by the neurologist Collin, localized the sympathetic synapses of the spinal cord and medulla.

W. H. Gaskell[20, 24] (1875-), Langelaan, and J. H. Langley defined the autonomic and sympathetic system and laid the foundations for the modern conception of the autonomic nervous system.

Cerebellum

Marie Jean Pierre Flowrens[2] (1794-1867) was professor of comparative anatomy at the University of Paris. In carrying out extensive experiments on the central nervous system, he analyzed the functions of the cerebellum and described cerebellar ataxia and demonstrated cerebellar and labyrinthine vertigo. He also studied effects of alcohol on locomotion and concluded that alcohol acted specifically on the cerebellum.

Among some of the neurologists who made important contributions are S. Weir

Mitchell, Frederick Tilney, Mills, Weisenberg, Holmes and Hunt.[24]

Of S. Weir Mitchell[7,24] (1829-1914) it is interesting to know that his father, a wise physician, said, "You are wanting in nearly all the qualities that go to make success in medicine. . . . You have brains enough but no industry." But S. Weir Mitchell gained world renown and wrote prolifically, published 246 articles, 119 of which were on neurology and in addition, many novels.

James Ramsay Hunt's[29] classic description of cerebellar dyssynergia was one of his works on the static and kinetic motility system in which the functions of the striatal system were also investigated. Not related but close by were his original studies on herpetic inflammations and his original description of geniculate ganglion neuroglia.

Conclusion

As one skims the surface of the contributors and contributions to the nervous system of man excluding nosological entities, the task becomes involved even in such a superficial and incomplete treatise. Many important men must be slighted even if one were to compose a manuscript in book size. However, one is struck with the basic grounding of the contributors, who for a great part were clinicians. They started a specialty which is still in its infancy and had practically no tools to work with.

We now have their contributions as a foundation, and in addition we have electronic aids such as electromyography, electroencephalography, accurate measuring and amplifying aids, arteriography, pneumo- and ventriculography and a host of drugs including antibiotics and radio isotopes. That insatiable curiosity which makes for a good doctor and scientist must still be propagated with these aids for the betterment of neurology and medicine.

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Neuropsychiatry In the Aleutian Islands

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Contrary to opinions held by many persons, military service in the Aleutian Islands is associated with an extremely low incidence of incapacitating mental illness. Burns[1] and Gelbman[2] have previously noted this fact. Both authors refrained from further comment however, preferring to deal with the etiological factors which were possibly operative in the men who did become psychiatric casualties. Although Burns discusses climate and other conditions peculiar to the Aleutian Islands, he states that intrinsic factors had an important role in the causation of mental disorders, as for practical purposes all of the soldiers were subjected to identical external factors. Gelbman expresses a strong dislike of the situation as it existed during his tour of duty as a neuropsychiatrist on Adak. He stresses the external factors as being major in the development of neuropsychiatric illnesses and in addition he lists a great many of the common gripes and resentments which have been universally voiced by soldiers in World War II and other wars.

The purpose of this paper is to report certain statistics concerning the neuropsychiatric non-effective rate of troops stationed on the Aleutian Island of Shemya and to discuss those factors responsible, in the opinion of one physician, for the unusually low rate. The report is based on observations made during 18 months service on the island from January 1945 to May 1946 as neuropsychiatrist and from March to July 1946 as Commanding Officer of the 329th Station Hospital.

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Neuropsychiatric statistics for the entire United States Army for parts of the above period have been computed by Appel[3] and may be used for a rough comparison. According to Appel, the daily neuropsychiatric non-effective rate for the entire Army from 1 January to 31 July 1945 was 6.1 per 1000 troops. During the same period on Shemya it was 1.07 per 1000 troops. In other words, an average of 0.6% of the total Army was in hospital or quarters on any one day as compared to an average of 0.1% of the troops on Shemya.

Of the 1,000,000 neuropsychiatric admissions to hospitals from 1 January 1942 through 30 June 1945 listed by Appel for the total Army, 7% were for psychosis, 64% for psychoneurosis and 29% for psychopathic personality and other conditions. From 1 January 1945 to 2 May 1946 there were 85 admissions of military personnel to the neuropsychiatric service of the 329th Station Hospital. Of these 41% were for psychosis, 41% for psychoneurosis and 18% for psychopathic personality and other conditions. A sharper contrast is seen with further analysis of the above figures. The average daily non-effective rate due to psychosis appears to have been essentially the same for Shemya as for the Army as a whole; 0.43 per 1000 troops for Shemya and 0.42 per 1000 troops for the entire Army. However, the non-effective rate due to psychoneurosis for the Army as a whole was nine times as great. The rate for the Army was 3.90 per 1000 troops whereas that for Shemya was a mere 0.43. Likewise, the non-effective rate for psychopathic personality and other conditions was nine times greater for the entire Army than for She-

mya. Compare 1.76 with 0.19 per 1000 troops.

In spite of the difficulty in working accurately with the above figures it does appear that 6,000 soldiers packed on one desolate base in the Aleutian Islands had an extremely low neuropsychiatric non-effective rate. An attempt to explain this apparent fact is therefore indicated. Many considerations are based entirely upon personal opinion and consequently are subject to as many reservations as one might care to make, just as issue can be taken with almost all of Gelbman's conclusions and recommendations.

One of the most important forces which tended to keep the neuropsychiatric non-effective rate low was the attitude held by the average soldier in respect to his service on Shemya. This attitude, peculiar to Aleutian soldiers, seemed to develop in direct proportion to the length of the time on the island. It will be understood better if Shemya, itself, and its development into a major base are first described.

The island of Shemya is a speck at the tip of the Aleutian chain, approximately forty miles from Attu. It is roughly diamond in shape measuring two miles by four miles and rising less than 500 feet out of the sea. Its smallness and its exposed position accentuate all of the disagreeable features of Aleutian weather. Tundra is the only vegetation.

United States troops made an unopposed landing there in the early summer of 1943 as soon as the outcome for the battle for Attu became predictable. They were soon followed by large numbers of civilian construction workers who rapidly altered the island's topography building a huge bomber runway and two fighter strips. The first winter was spent in tents and yak huts. During the second winter most of the soldiers and part of the civilians were housed in Pacific huts. By January 1945 there were approximately 6,000 soldiers and 4,000 civilians on the island. Although the population decreased following V-J Day to about 2,500 soldiers and slightly more than 100 civilians by January 1946, replacements brought the number of soldiers up to 4,500 by July 1946.

After the initial landing party, no person set foot on the island without being met with the universal greeting, "You should have been here when it was rough," and any complaint by a newcomer was automatically answered by the same remark. Variations of this theme were constantly reflected in the attitudes of the men toward each other. The early arrivals had accomplished much in spite of dismal living conditions and the Aleutian climate. But somehow they felt that they were being overlooked and forgotten by the folks back home because combat action on the island, other than for certain air corps troops, was potential rather than real. They noticed in the news from home that public attention was focused on the more active combat areas. Therefore, the men on Shemya were anxious to impress all newcomers that they had pioneered the island "when it was rough." Those who had "waded ashore" and rated a battle star on their theatre ribbon were particularly proud and by the same token they were extremely intolerant of any complaining new arrival. Personal prestige rested solely on the length of time a soldier had spent on the Aleutian chain. Thus a supportive type of group psychotherapy was in constant operation on a large scale. A replacement ceased to be such the moment he managed to corner a stranger to tell him he should have been on the island when it was rough.

The tour of duty for military personnel in the Alaskan Department not on flying status, was two and one-half years. Flying personnel, many of whom participated in long bombing runs over the Kurile Islands, were sent home after one year. During the first half of the 18 month period covered by this report, many men completed their tours, were replaced, and rotated to the United States for further duty. As a soldier couldn't be rotated until his replacement arrived, the length of duty tours varied a great deal. Some men left after as little as 24 months, while circumstances kept others for periods up to 36 months. Following V-J Day and the establishment of the point system, a gradually increasing number of young troops arrived to take the places of those who had become eligible for separation from the service. The latter went home for various reasons of eligibility and their

time in Alaska varied from a few days to approximately thirty months. However, at all times the personnel changed gradually so that the attitudes of those who were leaving were accepted and perpetuated by those who remained.

The average soldier on Shemya voiced very definite opinions about the position he occupied in reference to his fellow soldiers on the island and elsewhere. He was willing to remain there doing the best sort of job he could and putting up with all kinds of hardships as long as the next fellow had to do the same. He was very much opposed to the concept that unwilling and disinterested soldiers should be rewarded by dismissal from the service and a trip home. And as might be expected, much of the administration involved in the evacuation of neuropsychiatric casualties was looked upon with suspicion. The men thought that their service on the island was serious business. It was personally important to them. They were contributing a productive portion of their lives and as mentioned by Burns many of them felt that they were missing most of the glories which compensated service elsewhere. It is no wonder that they held a person's military obligation to be far more important than any concepts of social ideology. In contrast to the point of view prevalent in many training camps in the United States, it simply was not socially acceptable to be evacuated from the island with a mental illness. However unenlightened and unreasonable this attitude might have been, it may be regarded as a positive factor in keeping a number of soldiers with minor neurotic symptoms from seeking disposition through neuropsychiatric channels.

Gelbman comments that the presence of civilian construction workers on Adak had a devastating effect on the morale of the troops. Such was not true on Shemya. Actually, the men on Shemya considered the civilians there as making an unusual contribution to the war effort. However, the soldiers did regard the civilians as being somewhat strange for ever asking of their own free will to be sent to the island.

The harshness of the climate was a positive rather than a negative factor in promoting mental health. Gelbman implies the opposite in remarking, "One must have considerable incentive and fortitude to put on boots, clothing, heavy parka, then walk 100 yards in the wind and rain in order to empty the bladder, or take a shower, or clean the teeth." However, in responding to numerous daily necessities, men serving in the Aleutians strengthened many traits of character similar to those of incentive and fortitude. A very definite contribution is made to ego strength when one struggles through a williwaw to take a shower in a weatherproof tent and then returns to exclaim to his hut mates "By Gad, I made it! It sure is rough out."

The monotony of Aleutian service was in itself a stabilizing influence. There were only two periods of stress, the one immediately following a soldier's arrival on the island and the one immediately preceding his departure. The opinion that intrinsic factors were more important etiologically than extrinsic factors is supported by the fact that practically all of the psychotic reactions occurred during the first or during the last few months of a soldier's projected tour of duty. This was well demonstrated as the average time in months being spent there was gradually reduced. Symptoms might appear at 18 months in a soldier expecting to go home in 20 months, whereas they wouldn't appear until the 27th or 28th month in a man sweating out a full tour.

Incapacitating psychoneuroses were recognized in men with service on the island which varied from two to twenty months of which 46% occurred during the first six months. In no case of psychoneurosis requiring evacuation was normal rotation anticipated. At the time the man with 22 months was evacuated, he needed a minimum of eight months additional service in order to have been eligible for rotation. A few mild anxiety states were seen in some soldiers during their last few months of Aleutian service. These men often asked for sedation on sick call but were most anxious to avoid hospitalization. They didn't want to let anything interfere with their scheduled voyage home.

The military society which had grown up on the island was a very stable one. With the exception of frequent and sometimes

violent changes in weather, the average soldier's day was just about the same, month in and month out. Everyone had plenty of spare time. Within this stabilized society a soldier could make any one of a number of satisfying adjustments depending upon how much effort he might want to exert. But regardless of what a man might have wanted to do, there were no stragglers on Shemya. There was no place to which one could straggle. Thus the stabilized society existing on the island was in marked contrast to that of a busy training center or a combat situation.

The only recommendation which will be made in this paper concerns itself with the adaption of military personnel to isolated posts. As troops are rotated, replacement should not be made by M.O.S. (Military Occupation Specialty) except for certain technical classifications. Replacement by M.O.S. retards promotion of many ambitious, deserving soldiers. It is much easier for one to serve in a desolate spot if he feels he is making progress and receiving some recognition for his efforts. For the first nine months covered by this report practically all replacements were made by M.O.S. but during the last nine months on-the-job training was carried out. Such training was challenging to both officers and men. Promotions were rapid, duty performance was excellent and morale was high.

My personal attitude in serving as the island's neuropsychiatrist may have had little or nothing to do with the low non-effective rate but it is of interest for I was one of the "ninety-day wonders" turned out by the School of Military Neuropsychiatry at Mason General Hospital. Menninger[4] dismisses the contributions of a thousand or more of my contemporaries, whose major training consisted of the three months course at the school, with the remark that the intent was to assign these men to work only where there was supervision. Regardless of the high level intent, Gelbman and I were only two of a large number of young officers with less than a year's supervision who later found ourselves in neuropsychiatric assignments in isolated places completely on our own. Although novices in psychiatry, those of us who went to the school felt that we were experts in the philosophy of neuropsychiatric administration as advocated by its commandant, Col. William C. Porter, and which was belatedly adopted in March 1945 in W. D. Circular #81.[5] Also, most of us were very much impressed by the series of lectures given at the school by Capt. Herbert X. Spiegel.[6,7] One of his observations of the Tunisian campaign was that many soldiers did not become psychiatric casualties simply because they were not permitted to become such. Likewise, any of us who were subsequently assigned to large camps or staging areas were distressed by the ease with which many healthy soldiers were permitted disposition through neuropsychiatric channels.

The assignment on Shemya was approached with the point of view that the mission of a military neuropsychiatrist is essentially the same as that of all medical officers, namely, "To protect the fighting strength." It was felt then that a person should be kept on a duty status as long as performance was adequate or until his tour expired regardless of whether or not he was unhappy or neurotic.

The above point of view was fortunate for it proved to be identical with that held by the great majority of officers and men on the island. One explanation for this excellent state of affairs was later found in an early letter from the Commanding General of the Alaskan Department to the commanding officer of the island. Calling attention to the critical manpower situation in January 1944, the letter stated that it was incumbent on all commanders to make the best use of personnel available and one who permitted the discharge of an enlisted man in preference to making the necessary effort to place him properly and train him failed to meet command responsibilities. Also, the policy was established that no individual would be evacuated who did not require further hospitalization. This letter emphasizing the responsibilities inherent in the proper exercise of command continued in its effect long after its author had left the theater. It also anticipated the philosophy of W. D. Circular #81.

The pertinent portions of Section III of W. D. Circular #81 on personnel are worthy

of review as they were followed very closely during the last 15 months of the period covered by this report. Paragraph 2-a states, "The diagnosis of any type of psychoneurosis implies sickness and disability of some duration. It is not to be applied for reasons of expediency in order to effect a disposition. It will be applied only when its use is justified by the existence of a clinical picture which satisfies the criteria for psychoneurosis as established by good medical practice. The mere presence of psychoneurotic symptoms which do not significantly impair the individual's efficiency or the presence of a predisposition to psychoneurosis does not warrant the diagnosis of any type psychoneurosis. Such individuals, if otherwise sound, will be considered as having no disease."

Paragraph 2-e continues, "In determining the disposition of cases, it must be clearly understood that there are many causes for non-effectiveness other than sickness. Among these are inaptness, inadaptability due to emotional instability, lack of physical stamina, misassignment, defective attitude and unwillingness to expend effort."

Paragraph 2-g includes, "It should be clearly recognized that the presence of any type of psychoneurosis should not lead automatically to separation from the service. When an individual is suffering from a psychoneurosis which is not incapacitating, he will be returned to duty."

Among the conclusions in Paragraph 3 are the following: "The majority of the factors which determine the mental health of military personnel are functions of command. In other words, the main job of preventive psychiatry must be done by the commanding officer of the line. It is the responsibility of command to obtain maximum utilization of manpower by providing proper incentive and motivation and such reclassification, reassignment, rest, relaxation and recreation as exigencies of the military service permit."

Shemya proved to be an ideal place to carry out the principles of neuropsychiatric administration as outlined above, for there was only one psychiatrist assigned to the island and thus it was impossible for a person to shop for the cheapest psychiatric diagnosis which might lead to a discharge.

Most soldiers with neuropsychiatric complaints were evaluated as out-patients and were studied at length while continuing to perform duty. An active out-patient consultation service was maintained and the other medical officers on the island were encouraged to use it in all cases except emergencies. Most patients were admitted after out-patient consultation. Aside from the psychotic and the completely disabled neurotic any soldier who was inadvertently admitted directly to the neuropsychiatric service of the hospital without prior out-patient consultation, was quickly worked up and returned to duty for further evaluation on that status. The longest time spent in the hospital by a soldier who later returned to duty was 21 days; the shortest was two days while the average was 8.5 days.

Many of the medical officers assigned to the 329th Station Hospital were fortunate in that they also served as unit doctors in dispensaries. For over a year, in addition to other duties, I had the opportunity to hold sick call for approximately 1500 men in the port area. Therefore, psychiatric evaluation and treatment were available for a quarter of the island's troops at a dispensary level.

No record was kept of the number of men seen as out-patients on consultation, but 200 would be a conservative estimate. Most of them were seen about three or four times. Most stopped coming as soon as they could see that their complaints were not sufficient to warrant evacuation. It seemed that symptoms developed for a secondary gain often disappeared when the gain was not forthcoming. No estimate can be made of the great number of psychiatric problems evaluated on routine medical and surgical rounds as O.D., as hospital reconditioning officer, on sick call, etc.

Psychotherapy was offered to most neurotics and to some psychopaths. It was of an explanatory and supportive type and it did prove of value in anxiety states and certain somatization reactions. The psychotherapeutic interviews were based on the proposition that the soldier's symptoms were legitimate and justified but that they were of a temporary nature and could be

treated on Shemya as well as anywhere else. Usually the soldier was told that his symptoms were part of his emotional response to coming to Shemya and that he would become asymptomatic when he finally decided to make the best of a poor situation.

Regular interviews were scheduled as often as the patients would care to attend. Although the average soldier desiring treatment came 10 or 12 times, one out-patient came weekly for 14 months before being rotated home. Some psychotherapy was done over the telephone, calls being received almost any time of the day or night. Often patients would drop by the hospital for a chat after an absence of several months. As observed by Burns this was particularly true of psychopaths. It appears that the psychiatrist was about the only officer and symbol of authority with whom a psychopath could "blow off steam" and get away with it.

Only ten men were disposed of through administrative channels[8] during the 18 months period. Such disposition was recommended for a few others following study but certain company commanders did not want to initiate the proceedings as required by regulations. For the benefit of the morale of those soldiers who were trying to do a good job, the company commanders preferred to put up with some inadequate soldiers rather than reward them with a trip home. In one instance, a soldier for whom an administrative disposition had been recommended was court-martialed and sentenced to six months confinement. This was spent in a stockade made by barring off one end of a Pacific hut and his term wasn't credited to over-seas time.

On the basis of the observations made during the 18 months on Shemya, it is concluded that external factors had very little to do with the mental illnesses occurring in military personnel there. Also, it would be a mistake to regard all those evacuated as having "broken down" as a result of conditions peculiar to the Aleutian Islands. For a variety of reasons, some of them were no longer effective soldiers. Some were no different than when they had first come into the Army, a certain length of time being required to demonstrate a neurosis unresponsive to the normally effective motivating forces which operate within the military service.

Considering the success with which many neurotic soldiers were kept on a duty status on Shemya, it is gratifying to note that the current regulations on psychiatric standards for enlistment and induction state that a person with psychoneurosis of any degree will be acceptable if the psychoneurosis has not incapacitated in civil life.[9]

Summary

The following factors are listed in an attempt to explain why the neuropsychiatric non-effective rate of military personnel on one Aleutian island was one-sixth the rate of the United States Army as a whole.

1. The challenging environment produced a serious attitude and a deep sense of military obligation in the troops.
2. Command was exercised in an excellent manner.
3. A stabilized society was produced.
4. There was only one neuropsychiatrist on the island.

Author's Note: The opinions expressed herein are those of the author and are not necessarily concurred in by the Department of the Army.

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Insulin Shock Therapy

(A Reconsideration of Factors Responsible for Improvement)

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A re-evaluation of therapy in psychiatry is necessary as it is in all branches of the medical sciences. The percentages of patients improving with insulin shock therapy have been completely discussed in the literature, and it is evident that those physicians utilizing insulin shock therapy are convinced of its beneficial effects. The staff of this hospital is of the opinion that it is the treatment of choice in patients with schizophrenic reactions. However problems remain, namely the method by which insulin shock produces improvement and the type of patient best suited for the treatment.

It is obvious that only a limited number of patients with schizophrenic reactions can be treated in comparison with the total number of individuals suffering from this illness. Because so many hours in terms of personnel time are necessary and because at this hospital we are only able to devote 20 beds to the treatment daily five days a week, it is necessary to be discriminative in the choice of patients for insulin shock therapy. During 1949 a large percentage of patients admitted to this hospital were suffering from schizophrenic reactions. While it was often the general opinion of the examining physicians that certain patients would not benefit, nevertheless treatment was given upon the insistence of relatives or because the physicians desired to give the patient the benefit of every possible therapeutic endeavor. It was not uncommon for patients falling in such a category to receive 60 insulin shock treatments with no benefit.

One of the authors has known all the patients treated at this hospital with insulin shock therapy and has had a thorough knowledge of their immediate environmental stresses, thought content, and their responses. From September 1948 to March 1950, 100 patients completed insulin shock therapy, not including 20 patients who are currently being treated. Age varied from 19 years to 42 years. For purposes of comparison with other percentages the following figures are cited although it is not the purpose of this paper to give a statistical review of the duration of illness, age at onset or response according to category. The percentage of improvement at the hospital is believed to be very good because several years have elapsed since our patients were discharged from military service. It is probable that the onset in the majority of illnesses took place more than a year previously. It has always seemed futile to determine whether the onset occurred six months or two years prior to hospitalization or treatment because in the majority of instances where we have made an effort to determine the life situation of the patient we have found evidence of personality deviations being present many years before the actual onset of a frank psychosis; so that one cannot determine the actual time of onset.

Table 1 lists the number of treatment sessions received by our group of patients; the second table has to do with the number of hours of coma administered. Optimally, we desire to give patients 60 treatments or 20 to 30 hours of coma. One of the assisting residents initiated the plan of counting the hours of coma rather than the number of treatments. This is a practical plan because the benefit in the majority of patients seems to be related to the hours of coma, particularly in the more profoundly psychotic cases.

TABLE I

No. Sessions	*No. Patients*
50 to 63	42
40 to 50	23
30 to 40	19
20 to 30	4
Less than 20 treatments	12

From the Veterans Administration Hospital, Tomah.

TABLE II

Hours of Coma	*No. Patients*
30	17
20 to 30	41
15 to 20	12
10 to 15	9
Less than 10 hours of coma	21

A review of the patients of this group who have been placed on trial visit or discharged reveals that 48 patients have left the hospital and have not returned to date. Five more are eligible to leave in the near future, making a total of 53 patients who can be classified as greatly improved. This compares favorably with statistics from other series.

The question remains as to how we benefit patients with this type of treatment. The authors are of the opinion from their own observations and from the statements of patients that one major factor is a lessening of emotional tension, so that conflicts become less important, and as a result thought processes better organized. The individual is able to approach reality problems in a less emotional manner. It is believed that another factor making for improvement is the relief from emotional stress early in the course of treatment. Patients will often say that during the treatment unpleasant feelings are recalled which may be reflected in their facial expression and chance remarks although one cannot say that there is a true abreaction. Latent homosexual drives may be discovered. The thought content associated with the feeling is usually not recalled although one patient said that the old feelings of hostility towards others were recalled and the material seemed to be the same as might be found in a bad dream. It is not believed that thinking primarily becomes less complex, but that the better organization of thought processes is due to less interference from highly charged emotions. Often patients can give much dynamic material following a number of comas in therapeutic sessions without much analytic effort on the part of the physician; this probably occurs for the same reason, less feeling being attached to conflictive material. Added to these factors are those lesser ones of the high morale in the insulin clinic with group activity.

What are the characteristics of an individual who may respond to insulin shock therapy? One cannot say that only those with emotional turmoil may benefit and not those with an affect loss. While the latter type of patient does not respond, the agitated paranoid patient may not do so either. It has not been possible to reveal all the dynamic material in these cases of schizophrenia because of the nature of their illness; and one can not, with the limited time available and the large number of patients to be treated, make an attempt to foretell the outcome by means of such material alone. The majority of these patients revealed heterosexual maladjustment and this seemed to be the most common presenting conflict on the part of the patient. Hostility toward parents or siblings was common. A feeling of inferiority with a retreat from reality as might be expected was also commonly found. Homosexual trends were common. Feelings of guilt were not often found.

Prognostic factors such as pre-psychotic personality, acuteness of onset and presence of affectivity are not very satisfying to the physician who must use discriminative judgment in the selection of patients for insulin shock therapy. To the authors, one of the most outstanding characteristics of those patients who responded to treatment was that of having a well preserved personality. This might be called being "personable." Perhaps this is what one might expect when there is not a marked regression. This personality factor does not always hold true especially when there are marked feelings of hostility, but by and large it is a valuable prognostic evaluation. In an attempt to determine what we mean by being personable, we list a number of factors such as variety of interests in common with other people, favorable emotional responses to other people, a feeling of belonging to a group, an ability to meet others on a status of equality, feelings of independence as opposed to an excess of dependent feelings, and an ability to realize a need for assistance. A better picture of the personality structure may be revealed by amytalization. When there is an excess of regressive or dependent tendencies with associative withdrawal and lack of emotional response, the progno-

sis is poor. If an emotional response fluctuates and there is a marked dependency, the prognosis is not good.

An example of a type which may not be expected to respond to insulin shock therapy is that of one patient recently completing treatment. Upon admission he seemed to be mildly depressed so that he could not be said to lack an emotional response. He appeared passive but friendly. He stated that he thought that he was chained to women and that he was part woman and part man. At the time that the treatment was instituted, no clue could be found for these statements. After he had received 40 treatments and 25 hours of coma he was unimproved. He could, however, give more satisfactory information regarding his conflicts in the past. When he was five years old his mother died and his earliest memory is that of being near his mother's coffin and crying. He was raised by an older sister but he never found anyone to replace his mother's love. He seemed to introject mother's love because he stated that when he felt like a woman, it had to do with a feeling of love. He had had the same feeling when he was a child and longed for his mother. Being chained to women meant being chained to someone who loved you like a mother. This patient had been married and divorced. He could not find a mother substitute in the wife. It is obvious that he has had marked dependent and regressive longings since childhood. He has never felt as though he belonged to a group. He has always felt inferior and has never been able to meet others with a feeling of equality. His response to others emotionally was interfered with by his longing for maternal protective love and he has never had a variety of interests in common with others; instead of the feeling of independence, his has been one of marked dependency.

Discussion:

Other factors than those mentioned may be responsible for improvement in insulin shock treated schizophrenic reactions. The actual effect of insulin is unknown, but it is believed that a lessening of emotional stress —which may be partially due to a type of abreaction or possibly because of some effect upon the central nervous system—is the most important factor in making for improvement of the successfully treated patient. Undoubtedly the individual's ability to resolve conflicts is also of benefit and this occurs at times after treatment has progressed for a considerable period and as the patient improves.

The personality factors which we have mentioned are probably not complete but may be of some assistance in consideration of the selection of patients for treatment.

Conclusion:

1. A review of 100 insulin shock treated cases again reveals the value of this therapy for schizophrenic reactions.
2. The intactness of the personality is the best criteria in considering the prognosis of schizophrenic reactions, with marked dependency being an unfavorable indicator.
3. It is believed that relief from emotional stress is most important for the improvement of the patient; this occurs through the reproduction of unpleasant feelings during treatment, and possibly also because of some effect of coma upon the central nervous system.

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Neurological Clinical Pathological Conference

of the

Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

CASE NO. J. H.—A-1594

Presentation

A 68-year-old white man was well until about July 1950. An elective hydrocelectomy and left orchidectomy had been performed in February 1950. The reason for the orchidectomy was not apparent. Although he had suffered periodic epigastric distress for many years, relieved by bicarbonate of soda, there was no history of prior complaint.

About the 1st of July the patient's family noticed that his speech had become slightly slurred. On July 15 the patient ate his usual evening meal and then took a nap. In a short while he awakened with extreme dizziness. He arose from the chair in which he had been sleeping and immediately fell toward the left to the floor. He vomited his dinner and was unable to rise without falling to the left. He had never experienced such an attack before.

On July 16 the patient complained of pain in the left occiput. On examination he was found to be prostrate and appeared acutely ill. He was moderately obese, his face was flushed, and his skin cold and clammy. Temperature was 98.6 degrees F., respirations 20 per minute, blood pressure 210/110. The heart rate was 68; the rhythm was normal. The chest was emphysematous. Passive elevation of the head induced intense vertigo and leaning to the left. Rapid horizontal nystagmus accompanied attempted right lateral gaze. The deep reflexes were hyperactive, more so on the right. Local weakness was not demonstrable. Several beats of clonus could be elicited at the right ankle. Hoffmann's sign was present on the right, and there was an extensor plantar response on that side. Rapid alternating movements and the finger-to-nose and heel-to-shin tests were performed well bilaterally. Later that day the patient complained of a dull pain in the left chest and a feeling of fullness substernally.

Lumbar puncture showed an initial pressure of 145 mm. Ten cc. of clear fluid was removed and the final pressure was 130 mm. The fluid contained no cells; the total protein content was 63 mg. per cent. A Pangborn serological test on the cerebrospinal fluid was negative.

On the following day the patient experienced difficulty in swallowing. This symptom persisted during his remaining life. The pain in the left chest continued with variable severity, but gradually became more pronounced. At times there was pain in the left upper abdominal quadrant as well. Nausea and vomiting were present intermittently. Electrocardiograms on July 16, 17, 19, 21, and 26 showed only non-specific changes.

On July 17 the neurological signs were unimproved, and additional findings included limitation of upward gaze and diplopia on forward gaze. There was diminished appreciation of cold over the left side of the face and in the right extremities. A slight sucking reflex was present. The peripheral blood contained 6,700 leukocytes, 64 per cent of which were neutrophiles, 23 per cent lymphocytes, and 13 per cent monocytes. There was 17.3 mg. per cent of hemoglobin and 4.84 million erythrocytes per cubic mm. The erythrocyte sedimentation rate was 11 mm. per hour; a blood Kline test was negative.

On July 18 the patient was quite ill with the pain and severe vomiting. The vomitus contained occult blood. The heart rate remained regular at 120 per minute. The pulse was bounding. Blood pressure was 150/75. The abdomen remained soft and was not tender. A roentgenogram of the chest showed a diagonal line of increased density in the right lower lung field. The patient continued to do poorly.

On July 19 a blood count revealed a leukocytosis of 28,800 cells per cmm.; there were 91 per cent neutrophiles, 8 per cent lymphocytes, and one per cent monocytes. The hematocrit was 53 per cent, and plasma chlorides were 313 mg. per cent. A barium swallow with fluoroscopy and a flat plate roentgenogram of the abdomen revealed no abnormality.

The patient's general condition improved temporarily until July 26, although there was no improvement in neurological signs. Past-pointing with the left hand and ataxic performance of the left heel-to-shin test were noted on July 25. On July 26 the patient became worse, his sensorium clouded, and Cheyne-Stokes respirations appeared. His pulse was slow, 52 to 60 beats per minute, the pupils became constricted and reacted poorly to light. His temperature rose slightly and he began to cough bloody sputum. Leukocytosis of 24,500 cells per cmm. with 71 per cent neutrophiles was noted. Hemoglobin was 15 mg. per cent. The patient gradually became comatose. Shortly thereafter there occurred a generalized convulsion in which the head turned to the right. During this convulsion respirations became gasping and ceased.

Differential Diagnosis

Dr. John Campbell: For purposes of discussion, I have divided the problem into four headings, the localization of the acute cerebral episode, the reason for this episode, the complications, and finally the cause of death.

The history prior to the onset of the hy-

drocelectomy and the orchidectomy must be considered unrelated in the light of the final outcome, but the epigastric distress which suggests chronic gastric disease, peptic ulcer, or possibly malignancy must be considered later in discussing the complications. The only significant findings prior to the onset of the acute illness were obesity and hypertension. Dysarthria was present for two weeks, from July 1 to 15, before the onset of an acute attack of dizziness, nausea, and vomiting. Falling to the left, prostration, and hypertension were found on the initial examination, associated with the vertigo and falling to the left with elevation of the head. This latter sign might be called Bruns' syndrome; appearing alone, it suggests fourth ventrical tumor or Cysticercus cyst. The rapid nystagmus in right lateral gaze, focal but minimal pyramidal signs, and the pain in the left occiput pointed to a posterior fossa lesion or more specifically a lateral medullary lesion. This localization was further confirmed on the second hospital day by the onset of dysphasia (involvement of the motor nucleus of the 10th nerve), hypalgesia to cold on the left side of the face (involvement of the descending root of the 5th cranial nerve and its nucleus), with diminished cold appreciation on the opposite side, not involving the trunk or neck (spinothalamic involvement adjacent to the nucleus and the descending root of the 5th nerve). The continued nausea and vomiting so characteristic of medullary disease can be accounted for by involvement of the 10th cranial nerve and its dorsal motor nucleus in the medulla, most probably root involvement in the medulla. The subsequent findings of limited upward gaze and the diplopia may be seen in this lateral medullary syndrome. Parinaud's syndrome is, of course, more often associated with superior collicular disease, but medullary involvement such as by tumors of the cerebellar-pontine angle or the posterior fossa may be complicated by this finding. The eye signs are probably related to involvement of the posterior longitudinal bundle in the medulla. The sucking reflex found on the second or third day is not of localizing value and suggests more diffuse brain stem damage. Horner's syndrome was absent. No further neurological findings were noted until the day prior to death when left cerebellar findings completed the syndrome of the posterior inferior cerebellar artery.

To visualize the anatomical localization of this syndrome, we should look to the medulla in the mid-olivary region. The vascular supply of the lateral medulla is a branch of the posterior-inferior cerebellar artery. In the mid-line the pyramidal tracts, inferior oliva, medial lemniscus, posterior longitudinal bundle, and the 12th nucleus are supplied by the anterior spinal artery; just lateral to this, including most of the retroolivary area, the blood supply is via a branch of the vertebral artery. The restiform body may receive its blood supply from the posterior-inferior cerebellar artery or the posterior spinal artery, if the latter be present.

The structures so far involved have been on the left side and include the ascending root of the 5th nerve and its nucleus, the spinal vestibular nucleus, nucleus ambiguus, and roots of the 10th nerve from the nucleus ambiguus and the dorsal motor nucleus. The minimal pyramidal tract signs on the right (the opposite side), absence of proprioceptive findings and of weakness of the tongue, and the absence of Horner's syndrome (which may occur in lateral medullary lesion due to the involvement of sympathetic pathways in the reticular substance) further confirm this lateral medullary localization.

As far as the cause is concerned, differential diagnosis should include hemorrhage which is ruled out by the precise localization of the lesion, the absence of red cells in the cerebrospinal fluid, maintained consciousness, and the improvement which occurred during the course, followed by complications and death. There is no reason to suspect an embolism, the prodromal signs speak against this. There is no obvious point from which an embolus could have arisen. Primary tumor seems unlikely from the clinical course; a secondary tumor may, of course, involve this area. Meningovascular syphilis is ruled out by the normal serology both in the blood and cerebrospinal fluid. Polycythemia was ruled out by the normal red count. The hematocrit which

rose later I feel was due to dehydration.

Prodromal signs in thrombosis associated with arteriosclerosis occur in at least 50 per cent of cases, sometimes lasting as long as years, sometimes just weeks. The hypertension found on the admission examination, the obesity, normal cerebrospinal fluid, except for slight elevation of protein, all point to thrombosis due to arteriosclerosis. Usually this syndrome is not associated with such an unhappy outcome, improvement occurs after an onset with nausea and vomiting.

From the second day of this man's illness he had pain in the left chest, the left upper quadrant of the abdomen, associated with severe nausea and vomiting. This was a continued complication throughout the remaining illness and gradually became more severe and was associated with hematemesis, visceral signs of rapid pulse, blood pressure drop, and subsequently dehydration with depletion of chlorides. The physical findings as far as the chest and abdominal complaints were concerned were of no help in making a diagnosis. The abdomen was soft and not distended and not tender. Chest findings apparently were normal, and this was confirmed later by roentgen studies which revealed evidence of what was apparently an interlobar pleurisy on the right. The EKG studies which were done serially were non-specific, therefore, we can rule out a myocardial infarct.

It appears that those attending this patient were confused by the continued nausea and vomiting and were attempting to find something in the gastro-intestinal tract to account for it. The visceral symptoms at the onset can be explained by the vascular episode that occurred in the medulla, but for explanation of the continuation of these symptoms, one returns to the complaints of epigastric distress which this man had for years. One wonders about a peptic ulcer. There is no history to suggest malignancy; there was apparently no weight loss or other signs, but we must consider the possibility of obstruction associated with a gastric lesion brought on or aggravated by the nausea and vomiting which occurred as a result of the vascular episode. Uremia must have been considered as a possibility; I am sure it was ruled out. There was no evidence of interstitial pulmonary edema on X-ray, which further rules out the possibility of uremia.

Leukocytosis and hemoptsis signaled the onset of terminal bronchopneumonia. There was nothing to suggest pulmonary infarct as a terminal episode. The bronchopneumonia could be explained by aspiration, aided and abetted by dysphagia and the poor general condition of the patient due to his gastrointestinal complications. On the 10th day he developed further neurological findings. On the 11th day terminally he showed signs of cerebral edema as manifested by the slow pulse, respiratory distress and Cheyne-Stokes respirations, clouded sensorium, and convulsions. Even though there was suggested irritability of the left cortex, I don't feel it was important in indicating further thrombosis, hemorrhage, or metastatic lesion. Respiratory failure following the convulsion resulted in death.

My diagnosis in this case is: (1) Hypertensive cardiovascular disease with arteriosclerosis, cerebral arteriosclerosis with thrombosis of the posterior inferior cerebellar artery, (2) peptic ulcer or gastric malignancy associated with obstruction, (3) bronchopneumonia, and (4) cerebral edema.

Dr. Charles D. Aring: The students were in agreement with Doctor Campbell and rather unanimously thought that this was a lateral medullary syndrome. Not all of the students discussed the gastric manifestations. Some thought they may have indicated the reactivation of a peptic ulcer; one felt that it probably was a neurogenic ulceration of the Cushing type. I might say that Dr. Campbell was quite right in indicating our confusion about the development of this man's signs and symptoms. I saw him initially as a consultant. Certainly with experience one becomes optimistic as far as the immediate prognosis in lateral medullary syndrome is concerned. I thought this man, who was in relatively good shape when I first saw him with obvious signs of a lateral medullary syndrome, had a good prognosis. With the continued progression of signs, we made valiant attempts to come to a decision as to what was happening.

Pathological Discussion

Dr. Murton Shaver: The necropsy was performed about nine hours postmortem. The most important findings included marked dilatation of the stomach which contained about 300 cc. of blood. Superficial erosion of the inferior portion of the esophagus was also observed with hemorrhagic effusion into the mucous layer. A large quantity of blood was found in the small intestine, and a small ulcer without evidence of hemorrhage was found in the duodenum. The heart was dilated but not increased in weight, there being no gross hypertrophy of the muscle wall.

The brain weighed 1400 grams. The cortical veins were congested, but otherwise the superficial appearance was not remarkable. The larger vessels of the circle of Willis showed marked arteriosclerotic change, and it was observed that the left vertebral artery was almost as large as the basilar artery. Both of these vessels showed a great degree of sclerotic change, but no obstruction was found in their lumina. Softening was found in the left dorso-lateral portion of the medulla and in the inferior surface of the left cerebellar hemisphere. A small area of softening was also found over the superior surface of this hemisphere. Serial sections through the brain confirmed the locale of the areas of softening, and no other gross abnormality was found.

Microscopic sections made through the medulla at the level of the inferior olive demonstrated a somewhat wedge shaped area of softening extending dorsally from the posterior portion of the olive to the region of the spinal tract of the trigiminal nerve and its nucleus. In this region the parenchymal structures were unrecognizable and the tissue had a fragmented appearance. There was an early increase in endothelial cells in this region while the blood vessel walls showed some fibrous thickening in the smaller arteries and arterioles with occasional small extravasations of red blood cells in the perivascular spaces. A few lymphocytes were found occasionally in these spaces. Medial to the area of softening the cells of the hypoglossal nucleus and the dorsal motor nucleus of the vagus were partially preserved, but in this region a few small perivascular extravasations of red blood cells were seen although the surrounding tissue appeared fairly well preserved. No point of vascular occlusion was seen in any of the vessels in these sections.

Sections made through the cerebellum revealed scattered areas of recent and older infarction. In some of these, large numbers of compound granular corpuscles or histocytes were scattered throughout the tissue which was fragmented with loss of nerve cell structure. In other areas the tissue appeared fairly well preserved except that it stained more deeply than usual, and a few perivascular lymphocytes were seen with occasional perivascular petechia, while the nerve cells and axons appeared swollen and distorted. Careful inspection of the vessels in these sections fail to reveal any point of occlusion.

Sections made through the left vertebral artery demonstrated the large size of the vessel and showed the very marked arteriosclerotic change described grossly in the vessel wall. The lumen was markedly narrowed, but there was no evidence of occlusion.

Sections through the lower esophagus demonstrated the diffuse infiltration of red blood cells into the mucous layer and loss of the overlying mucous membrane. There was some generalized necrosis of the tissues invaded, but no single large vessel seemed to be eroded in any of these sections.

As I mentioned, there was marked dilatation of the stomach and some dilatation of the heart. It is thought that these are associated with failure of vague innervation. If, on the other hand, the vagus is stimulated excessively, it is thought to cause excavation and ulcer formation. Bleeding in this case apparently was not from any single large vessel, but was from a diffuse effusion extending throughout the lower area of the esophagus. This sort of hemorrhage is thought to occur only in autonomic imbalance when the sympathetic is intact. In this patient you will recall the absence of Horner's syndrome. In experimental animals stimulation of the anterior hypothalamus and tuber cinereum will produce this type of hemorrhagic lesion only when the

sympathetic system is intact, sympathectomy protects the animal. This case presents an interesting indication of the function of the autonomic tract as it passes down through the medulla; in this case there has occurred imbalance, with death occurring from massive hemorrhage in the gastrointestinal tract.

Clinical Diagnosis

Lateral medullary syndrome due to thrombosis of left posterior inferior cerebellar artery.

Doctor Campbell's Diagnosis

Hypertensive cardiovascular disease with arteriosclerosis.

Cerebral arteriosclerosis with thrombosis of the posterior inferior cerebellar artery.

Peptic ulcer or gastric malignancy associated with obstruction.

Bronchopneumonia.

Cerebral edema.

Anatomical Diagnosis

Encephalomalacia of focal disseminated type in the lateral medulla and in the region of the distribution of the posterior inferior cerebellar artery to the cerebellum due to arteriosclerosis.

Hemorrhagic ulceration of lower esophagus due to autonomic imbalance resulting from the lateral medullary lesion with terminal massive hemorrhage and death.

Student: Did the heart show any suggestion of infarction?

Dr. Shaver: No. The coronary vessels were diminished in calibre, but patent.

Dr. Aring: This type of upper gastro-enteric ulceration is a problem that concerns neurosurgeons; it is an occasional complication of brain operations particularly in the posterior fossa. Cushing surveyed this subject and found that this combination had first been reported by Rokitansky in 1841. Cushing reported some eleven cases from his rich material; nine or so of these were cerebellar tumor or cerebellar pontine angle tumor, presumably involving the autonomic fibers as they descend in the pons and medulla from their origin in the diencephalon. One of Cushing's cases was a hypothalamic tumor, and another, as I recall, was afflicted with hypertensive encephalopathy with diffuse lesions in the central nervous system. Anyone who is particularly interested in this syndrome should consult Cushing's monograph[1] which is contained in his papers on the pituitary body and the hypothalamus.

Student: According to the pathological report there was no thrombosis. I'd like to know how common it is to have an acute onset such as occurred here without gross obstruction to the blood supply.

Dr. Aring: I think the answer to that question depends largely upon the care with which the material is examined. Obviously, it is possible for occlusions of small vessels to escape the attention of the pathologist. Generally, it has been our experience to have happen just what happened here. When the diagnosis of cerebral thrombosis is made clinically, we are frequently unable to demonstrate complete occlusion of vessels either grossly or histopathologically by the techniques that are available to us. Some of us have come around to thinking that certain dynamic changes occur in circulation and are capable of producing syndromes of cerebral softening without thrombosis occurring. In other words, vasospastic conditions may occur—we know with certainty that they do in migraine for example—and these sometimes result in permanent neurological damage. We suspect that this is the state of affairs in hypertensive brain disease.

There are, of course, other methods of incurring local softening of the brain without thrombus formation. One of these is inefficiency of the heart. The ineffectual heart cannot adequately supply a tissue that at all time requires adequate blood. It is then possible for softening to occur in areas supplied by vessels that are thickened. It is well-known clinically that in not a few instances, coronary occlusion first makes itself known by a hemiplegia, the so-called cerebral debut of coronary occlusion. The unwary physician may overlook the damage in the heart and think that the primary cause of the patient's difficulty is cerebral, when, as a matter of fact, the cerebrum is only reflecting the inefficiency of the heart.

So, when a lessened blood supply is delivered to the brain, particularly to areas supplied by arteries that are burdened by sclerosis, I believe the possibility exists for

softening to occur without thrombosis. As I said before, we have not been able to find thromboses in vessels with the frequency that we should expect to find them. Perhaps, as Fisher[2] has recently suggested, a more thorough examination of the carotid circulation in the neck may clarify this problem.

Student: I missed how Dr. Campbell explained the limitation of upward gaze.

Dr. Campbell: It occurs with superior collicular involvement as with a pineoloma or when edema involves the colliculi. In this case we'd have to explain it as well as the pyramidal tract signs by invoking edema or some distance effect, for example, neural shock.

Dr. Aring: The protocol noted the upward excursion of the eyes as limited; it was not paralyzed. I personally don't believe that this is an unusual finding in very ill or aged people. It seems to me from a rather wide clinical experience in examining eye movements that it requires less effort to shift the eyes to the sides than upward. You can try it yourself. You rarely use upward excursion of the eyes, whereas you are constantly manipulating your eyes from side to side. I think it a fact that patients who become very ill don't elevate their eyes as well as they move them laterally, and I would surmise that this sign in this particular patient had no localizing value.

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Electroshock Therapy In the Presence of Pulmonary Tuberculosis

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We have been unable to find in the literature the report of a single case of a patient with pulmonary tuberculosis who was given pneumothorax and curare preceding each electroshock treatment. The following report is of such an instance.

Case Report

S. S., age 52, began to cough during the winter of 1947. Several months later his cough became increasingly productive and on occasion bloody. A roentgenogram of the chest revealed pulmonary tuberculosis and the sputum was found to contain acid-fast organisms. The patient was admitted to a sanatorium during March 1948. Examination soon after admission revealed exudative and productive tuberculosis involving the spaces of both lungs. There was a cavity in the first right interspace. Because of the persistence of acid-fast organisms in the sputum, pneumothorax was induced on the right side during May 1948. This was followed by sputum conversion on concentrate, but the cultures continued to reveal acid-fast organisms.

In July 1948 the patient became apprehensive and depressed, somewhat restless and frightened. He lost interest in everything and expressed self-destructive ideas. He had difficulty with concentration and worried inordinately about his lung affection. He also expressed numerous other somatic complaints referring to his stomach, feet and chest. His sleep was poor, his appetite had become worse and he had lost weight during the six weeks prior to admission to the mental institution.

He received seven electroshock treatments, six in the hospital and one extramurally. Grand mal seizures were noted during each treatment. To decrease the intensity of the muscular contraction, the patient was also given curare immediately preceding each electroshock treatment. In addition, the patient was given pneumothorax refills by Dr. Eli H. Rubin prior to each shock treatment. The refills consisted of approx-

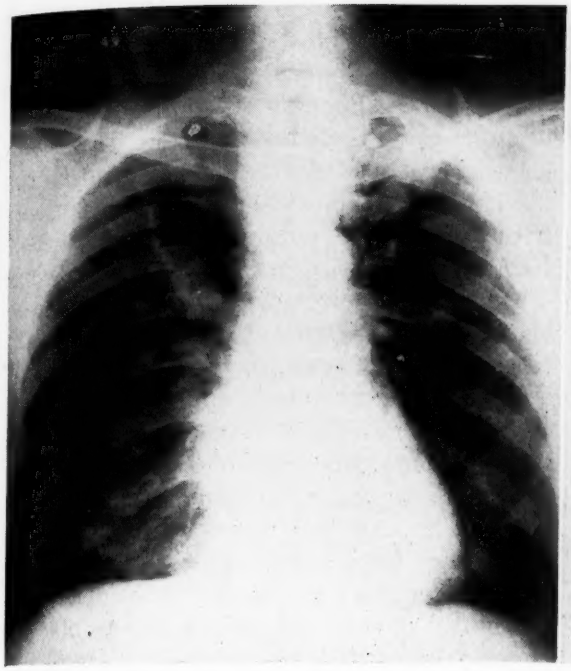

Fig. 1. Right lung is partially collapsed by a pneumothorax; adhesions suspending right upper lobe to chest wall. Fibrotic infiltrations left upper lobe.

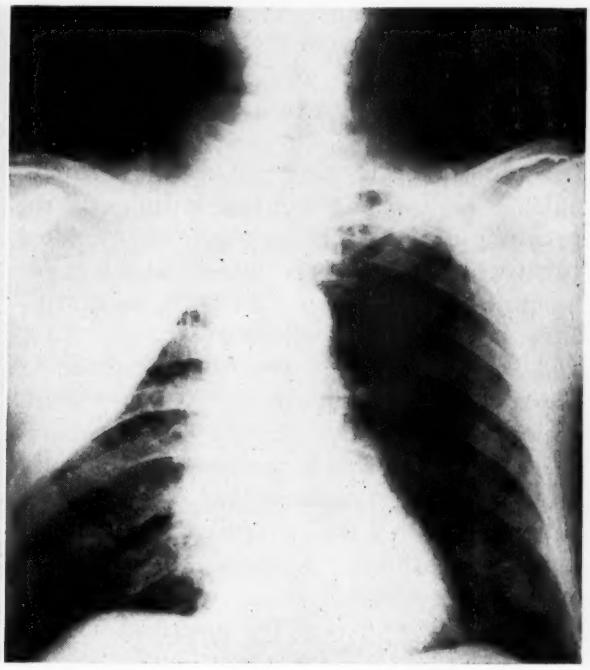

Fig. 2. Thoracoplasty collapsing right upper lobe.

imately four to five hundred cc. of air, the final pressure remaining near zero. During the period of shock treatment he had frequent fluoroscopic and x-ray examinations of the chest (Fig. 1).

The depression cleared up after the seventh treatment. Because of the presence of an adhesion suspending the right upper lobe and preventing complete collapse of the lung, a partial pneumonolysis was done during September 1948. Following this, he continued to receive pneumothorax treatments for more than a year but acid-fast organisms continued to appear periodically in the sputum. It was therefore decided to allow the lung to re-expand and to complete the collapse of the lung with thoracoplasty. In October 1949, a first stage thoracoplasty was performed by Dr. Morris Rubin, and segments of the third, fourth and fifth ribs were resected subperiosteally including small portions of the transverse processes. Two weeks later he underwent a second-stage thoracoplasty at which time the remainder of the third rib as well as the first and second ribs, including portions of the transverse processes were resected. An adequate collapse was obtained (Fig. 2). In spite of the surgical ordeal, there was no recurrence of depression.

Following discharge from the hospital the patient continued his convalescence at home. The disease underwent arrest, the thoracoplasty having been effective in converting the sputum so that at present no acid-fast organisms are obtainable on concentrate and culture. The patient returned to part-time work in May 1950.

Discussion

It is generally agreed that the presence of active pulmonary tuberculosis is a contraindication to the use of metrazol convulsion therapy. Bulley and Greene[1] noted that 25 of 227 schizophrenic patients (8.3%) treated with metrazol convulsive therapy developed pulmonary tuberculosis as compared with 3% of the untreated schizophrenics. There is a difference of opinion, however, regarding the use of electroshock therapy. A few investigators have treated such patients with shock treatment successfully; the majority are reluctant to undertake the treatment for fear of causing an acute flare-up of the pulmonary tuberculosis.

The literature on the subject is rather meager and contradictory. Hemphill[2] noted

activation of pulmonary tuberculosis in two patients treated with electroshock. It occurred within three weeks of the beginning of the shock treatments. One of his patients died within six months of military tuberculosis. The reactivation of the tuberculosis is ascribed by the author to the breaking down of fibrous tissue surrounding tuberculous foci by the violent chest movements and the postconvulsive hyperventilation incident to the shock treatment. Will and Duval[3] described a patient who had an apparently latent pulmonary tuberculosis which became active shortly after a course of eight electroshock treatments. Norman and Shea[4] noted reactivation of pulmonary tuberculosis in 3 out of 266 patients treated with electroshock. Kaldeck[5] reported a 20 year old psychotic patient with pulmonary tuberculosis who received electroshock and collapse therapy (pneumothorax) during the same period without any ill effect on his lung condition. On the other hand, he cited two patients who, three weeks and six months after completion of electroshock therapy, developed rapidly progressive pulmonary tuberculosis from which they died shortly afterward. Kaldeck stresses the fact that a certain number of chronic schizophrenics may be expected to develop pulmonary tuberculosis. This makes it difficult to evaluate the role of shock therapy in the reactivation of tuberculosis.

At a symposium on electroshock therapy, Zeller[6] expressed the opinion that tuberculosis, irrespective of location or stage, is a contraindication to electroshock treatment. At the same meeting Jones[6] however, reported a patient with active pulmonary tuberculosis who received electroshock for his mental condition and did not show any spread of his tuberculosis. Kleinschmidt and his co-workers[7] tell of a 38 year old man who had bilateral pulmonary tuberculosis with pneumothorax on the left side which required refilling at intervals. This patient was treated for a depression and attempted suicide. He received 11 electroshock treatments, 8 of which resulted in grand mal seizures. His depression cleared up entirely and no reactivation or extension of the tuberculous lung process was noted. Unfortunately, no control x-rays were mentioned in their report. Close[8] reported eight psychotic patients (mostly schizophrenic) with associated pulmonary tuberculosis who received electroshock treatments without any ill effect upon their lung ailment. However, he warned that "electroshock should not be used indiscriminately in psychotic patients with associated pulmonary tuberculosis." Lambiotte and co-workers[9] advised the use of curare with electroshock therapy of psychotic patients with associate pulmonary tuberculosis. Pacella,[10] after reviewing 300 shock-treated patients, mentions five who had a history of active pulmonary tuberculosis at some time within a fifteen year period prior to the institution of electroshock treatment. All had received routine treatment for their tuberculosis. One of the patients had, in addition, a therapeutic pneumothorax during the period of the electroshock treatment. All five showed improvement of the lung disease concomitant with the mental improvement. Jentoft[11] believes that pulmonary tuberculosis is no contraindication against convulsive treatments, even though in 3 of his 56 tuberculous mental patients, electroshock therapy aggravated the existing lung condition.

Summary

A man of 52, while receiving pneumothorax treatment for active pulmonary tuberculosis, received seven electroshock treatments for an endogenous depression. Prior to each treatment, he was given intravenous curare and an instillation of air in the chest. His depression cleared up quickly. Because the pneumothorax was technically unsatisfactory, it was discontinued and a two-stage thoracoplasty substituted. This was accomplished successfully and the patient eventually returned to work.

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